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Microwave Radiation (2450 MHz) Alters the Endotoxin-Induced Hypothermic Response of Rats

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16. ABSTRACT

The parental administration of bacterial endotoxin to rats causes a hypothermia that is maximal after approximately 90 minutes. When endotoxin-injected rats were held in a controlled environment at 22°C and 50% relative humidity and exposed for 90 minutes to microwaves (2450 MHz, CW) at 1 mW/cm , significant increases were observed in body temperature compared with endotoxin-treated, sham-irradiated rats. The magnitude of the response was related to power density (1 mW/cm^2>5mW/cm^2>1mW/cm^2). Saline-injected rats exposed for 90 minutes at 5 mW/cm^2 (specific absorption rate approximately 1.0 mW/g) showed no significant increase in body temperature compared with saline-injected, sham-irradiated rats. The hypothermia induced by endotoxin in rats was also found to be affected by ambient temperature alone. Increases in ambient temperature above 22°C in the absence of microwaves caused a concomitant increase in body temperature. This study reveals that subtle microwave heating is detectable in endotoxin-treated rats that have an impaired thermo-regulatory capability. These results indicate that the interpretation of microwave-induced biological effects observed in animals at comparable rates and levels of energy absorption should include a consideration of the thermogenic potential of microwaves.

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Microwave Radiation (2450 MHz) Alters the Endotoxin-Induced Hypothermic Response of Rats

R. J. Smialowicz, K. L. Compton, M. M. Riddle, R. R. Rogers, and P. L. Brugnolotti

Experimental Biology Division, Health Effects Research Laboratory, US Environmental Protection Agency, Research Triangle Park, North Carolina

The parenteral administration of bacterial endotoxin to rats causes a hypothermia that is maximal after approximately 90 minutes. When endotoxin-injected rats were held in a controlled environment at 22 °C and 50% relative humidity and exposed for 90 minutes to microwaves (2450 MHz, CW) at 1 mW/cm², significant increases were observed in body temperature compared with endotoxin-treated, sham-irradiated rats. The magnitude of the response was related to power density (10 mW/cm² > 5 mW/cm² > 1 mW/cm²). Saline-injected rats exposed for 90 minutes at 5 mW/cm² (specific absorption rate approximately 1.0 mW/g) showed no significant increase in body temperature compared with saline-injected, sham-irradiated rats. The hypothermia induced by endotoxin in rats was also found to be affected by ambient temperature alone. Increases in ambient temperature above 22 °C in the absence of microwaves caused a concomitant increase in body temperature. This study reveals that subtle microwave heating is detectable in endotoxin-treated rats that have an impaired thermoregulatory capability. These results indicate that the interpretation of microwave-induced biological effects observed in animals at comparable rates and levels of energy absorption should include a consideration of the thermogenic potential of microwaves.

Key words: endotoxin, hypothermia, rats, 2450 MHz, microwaves, heating

INTRODUCTION

With the ever-expanding use of radio-frequency (RF) radiation over the past 30 years, public awareness and concern has grown. Concern for potential health hazards to human beings from RF radiation has fostered much scientific inquiry (for recent reviews see Shils and Skolnik [1979] and Gandhi, [1980]). Heating by RF radiation at high intensities can cause biological changes by whole-body hyperthermia or by altered thermal gradients

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Address reprint requests to R.J. Smialowicz, Experimental Biology Division, Health Effects Research Laboratory, US Environmental Protection Agency, Research Triangle, NC 27711.

within the body [Johnson and Guy, 1972]. However, there have been reports of effects at low intensities of RF radiation without evidence of increased temperature [Baranski and Czerski, 1976]. A debate persists in the scientific community concerning the potential of RF radiation to cause biological effects by other than thermal mechanisms. Consequently, many attempts have been made to determine if such radiation can produce biological effects at intensities below a temperature-increasing level.

Recently, it has been shown that under certain conditions experimental animals exposed to microwaves at levels at or below 10 mW/cm^2, the recommended guideline of the American National Standards Institute [ANSI, 1974], display significant changes in biological activities. This has been particularly true for behavioral and thermoregulatory responses to microwave radiation at levels that apparently do not cause an increase in body core temperature [Thomas et al, 1979; Stern et al, 1979; Adair and Adams, 1980]. The present study shows that low-intensity ($\leq$ 10 mW/cm^2), continuous-wave (CW), 2450 MHz microwave radiation causes a significant increase in the colonic temperature of rats made hypothermic by bacterial endotoxin.

Lipopolysaccharide or endotoxin from Gram-negative bacteria induces a febrile response in man and several other species (Cooper et al, 1979). However, endotoxin administered parenterally to rats, guinea pigs, and mice induces hypothermia [vanMiert and Frens, 1968; Feldberg and Saxena, 1975]. This response, at low ambient temperature, is believed to be due to an interference by endotoxin with the control of heat loss which supersedes the hyperthermic response of small rodents because of the large surface area to body mass ratio [Bligh, 1973]. In support of this hypothesis, it has been shown that endotoxin-induced hypothermia in rats is affected by ambient temperature [Szekely and Szelenyi, 1979]. Furthermore, endotoxin-injected mice in an environment at 30 °C maintained normothermia, while those at a temperature of 15 °C were hypothermic [Berry, 1966]. We took advantage of this phenomenon and report here that low-intensity microwave radiation induces measurable heating in endotoxin-induced hypothermic rats.

MATERIALS AND METHODS

Experimentally naive 90-day-old male rats with a mean weight of 413 ± 34 g (CD, Charles River Labs, Wilmington, Ma.) were used. Rats were maintained on a 12:12 light-dark cycle and allowed food and water ad libitum.

Rats were injected via the saphenous vein with Salmonella typhimurium W lipopolysaccharide (LPS, Difco Laboratories, Detroit, Mi.) suspended in pyrogen-free saline. The LPS was boiled for one hour prior to use. All test tubes, syringes, and needles used were sterile, disposable, and pyrogen free. Immediately following injection, each rat was placed in a vented acrylic restrainer (Figure 1), and the colonic temperature was measured via a YSI thermistor probe (Yellow Springs Instrument Co., Yellow Springs, Ohio, No. 401) inserted 8.0 cm beyond the anal sphincter. The thermistor probe was lubricated with K-Y lubricating jelly (Johnson and Johnson, Inc., New Brunswick, N.J.). Temperatures were read to an accuracy of within 0.1 °C from a YSI Model 46 Telethermometer after they had remained stable for 15 seconds.

Four experiments were performed. The first examined the effect of LPS dose (2.5, 10, and 40 μg) on endotoxin-induced hypothermia in rats over time. LPS-injected rats in individual restrainers were placed in a controlled environment at 22 ± 0.5 °C and

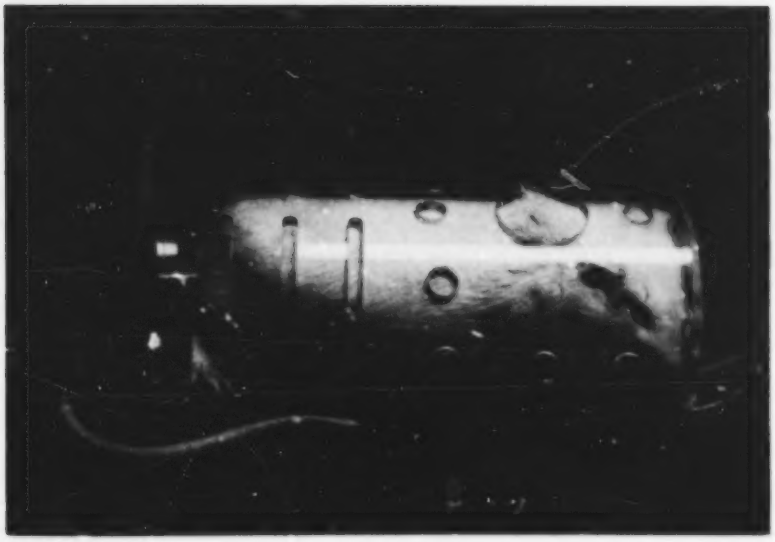

Fig. 1. Cylindrical acrylic restrainer. The area occupied by the rat is 17 cm long and 8.3 cm in diameter. The top of the restrainer has numerous 1.0 cm holes for ventilation.

50 ± 10% relative humidity. A thermistor probe was inserted into the colon with the probe lead taped to the base of the tail. Temperatures were continuously monitored over a period of 2.5 hours and recorded every 30 minutes.

The second experiment examined the effects of microwaves on LPS-induced hypothermia. Irradiations were conducted in a 2450-MHz exposure facility in which temperature, humidity, and air flow were controlled [Elder and Ali, 1975]. The microwave field was generated by a Varian Model PPS-2.5 AS industrial heating unit at a frequency of 2450 MHz in a continuous-wave mode. A total root-mean-square (RMS) ripple of 11% was measured after the installation of a full-wave bridge in the filament circuit. A controlled environment exposure chamber (61 × 76 × 76 cm) constructed of foamed polystyrene was positioned beneath a 10-dB-gain pyramidal horn antenna in the far field.

The environmental chamber was placed in an anechoic chamber that was lined with microwave-absorbing material (Model CBV24 and VCVB24, Emerson and Cuming, Inc., Canton, Mass). An environmental control system was used to regulate temperature and humidity in the exposure chamber and in an identical foamed polystyrene sham-irradiation chamber located outside of the anechoic chamber. Air temperature was maintained at 22 ± 0.5 °C, relative humidity at 50 ± 10% and air velocity at 0.31 ± 0.07 m/sec for all microwave and sham-irradiation exposures. A thermo-anemometer (Alnor Instrument Co., Niles, Ill) was used to measure air velocity at the center and at the four positions where the rats were positioned in the exposure chamber.

Following injection of LPS (40 μg) or saline, rats were placed in each of four positions in the exposure chamber 2.74 m beneath the horn antenna. The rats were placed at the points of a diamond-shape array at an equal distance (19 cm) from each other and 9.5

cm from the center of the chamber. The long axis of each rat was oriented parallel to the H-field vector with the head facing the air inlet port. Another group of four rats was positioned in a similar array in the sham-irradiation chamber. Rats were irradiated for 90 minutes at a power density of 10, 5, or 1 mW/cm^2. Power density was determined in the absence of animals and restrainers using an E-field dipole probe (Elder and Ali, 1975) placed in the center of the chamber at the plane in which animals would be irradiated. Power density was also measured at the four positions in the absence of restrainers and at each position with the other three occupied by a water load (350 g). No difference in the mean power density at the four positions was observed in the presence or absence of the water loads (8.3 ± 1.5 and 7.8 ± 1.1 mW/cm^2, respectively).

To randomize the microwave irradiations, only one group of rats was exposed to a given power density on each day. On the following day a different power density was used, and so on. Sham-irradiations of rats injected with LPS were also randomized so that a sham group was run simultaneously with an irradiated group on at least two occasions for each power density. All irradiation and sham-irradiation treatments were performed between eight and ten A.M.

The specific absorption rate (SAR) for the rats used in this study was approximately 2.0 mW/g at 10 mW/cm^2. This SAR value was obtained by twin-well calorimetry performed in this laboratory and previously reported by Kinn [1977] and Smialowicz et al [1979].

The third experiment examined the effect of procedural acclimatization on the response to microwaves. Six groups of four rats were acclimatized for a period of two weeks prior to treatment with endotoxin and microwaves. Each day during the acclimatization period the rats were handled in a manner similar to that required for endotoxin injection and microwave exposure. Rats were placed in restrainers, colonic temperature was measured, and rats were then placed in a controlled environment at 22 °C and 50% relative humidity for 90 minutes. At the end of 90 minutes, colonic temperature was again measured, and animals were returned to their home cages. After two weeks acclimatization to this regimen groups of four rats were injected with 40 μg of LPS and exposed at a power density of 0.5 mW/cm^2 for 90 minutes. A sham-irradiated group was always run simultaneously with the irradiated group.

In the fourth experiment, the effect of ambient temperature on LPS-induced hypothermia was examined. In this experiment, LPS-injected rats were maintained at ambient temperatures of 18, 22, 26, 30, or 34 °C, while their colonic temperatures were monitored over a period of 2.5 hours.

Data were analyzed using Student's *t*-test or Dunnett's *t*-test [Dunnett, 1955].

RESULTS

Figure 2 shows the time- and dose-dependent mean temperature of endotoxin-injected rats maintained in an environment at 22 °C. At LPS doses of 2.5, 10, and 40μg the nadir of the hypothermic response occurred at approximately 90 minutes. Therefore, we chose to measure colonic temperature at this time in experiments involving microwave irradiation. An LPS dose of 40 μg per rat, or approximately 100 μg/kg, was chosen for subsequent experiments. This dose is well below the LD_{50} for LPS in rats, which we found to be approximately 1.6 mg/kg.

Figure 3 shows the mean colonic temperature of rats exposed to 2450-MHz microwaves for 90 minutes. A body temperature response of endotoxin-injected rats that was related to power density is apparent. At each power density, the mean colonic temperature

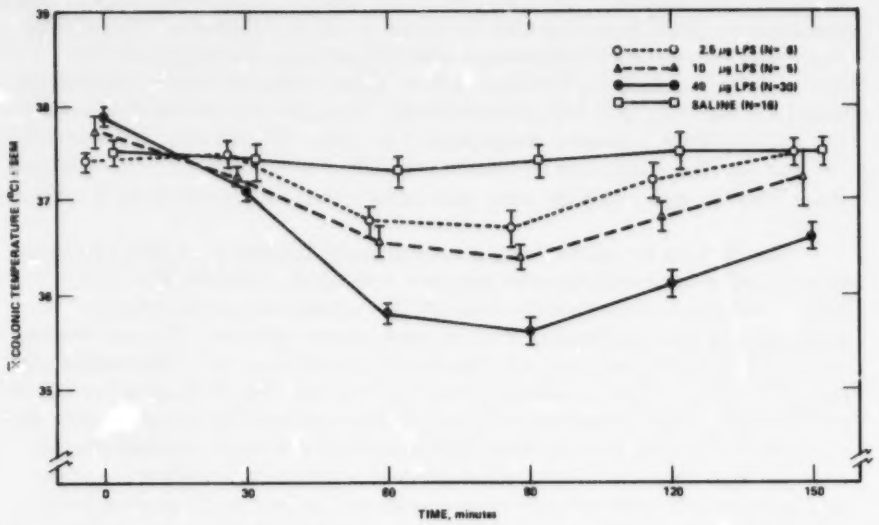

Fig. 2. Time and lipopolysaccharide (LPS) dose-dependent temperature response of rats to endotoxin (ambient temperature = 22 °C).

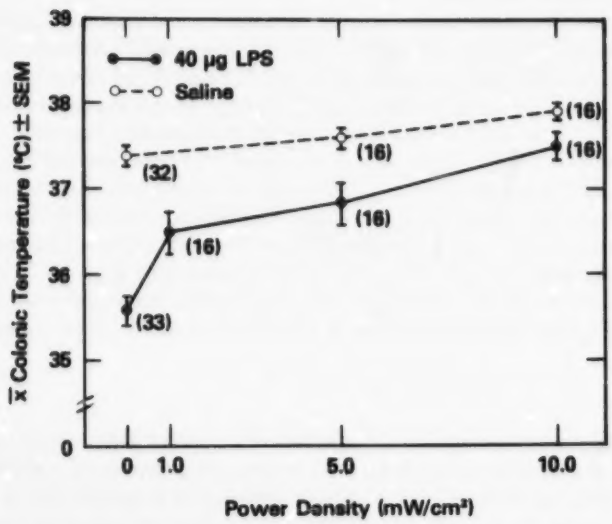

Fig. 3. The effect of 90 minutes of microwave irradiation on endotoxin-induced hypothermia in rats. The number of animals per treatment is shown in parentheses.

was significantly greater ($P < 0.05$, Dunnett's t-test) than that of endotoxin-injected, sham-irradiated rats. The difference between the change in colonic temperature (ΔT), ie, final temperature (90 min) minus initial temperature (immediately following endotoxin injection), for microwave-irradiated and sham-irradiated, endotoxin-treated rats were also statistically significant ($P < 0.05$, Dunnett's t-test). The mean ΔT's for the sham and the 1, 5, and 10 mW/cm^2 treatment groups were −1.6, −0.2, −0.5, and +0.1 °C, respectively. The 1 mW/cm^2 group had a smaller temperature difference compared with the 5 mW/cm^2 group. This was due to a slightly lower mean initial colonic temperature in the 1 mW/cm^2 group.

Table 1 shows the results from rats acclimatized to handling procedures two weeks prior to LPS injection and microwave exposure. A significant difference ($P < 0.05$, Student's t-test) was observed in the mean ΔT (°C) between microwave- and sham-irradiated, endotoxin-injected rats. However, there was no significant difference observed between the final (90 min) colonic temperature of irradiated rats and sham-irradiated rats despite the higher colonic temperature of the irradiated rats. This discrepancy in results is due to a slightly higher initial temperature of the sham-irradiated rats compared with that of the microwave-irradiated rats. While these results are inconclusive, they indicate that rats made hypothermic with endotoxin may exhibit a temperature response at a power density as low as 0.5 mW/cm^2 after an appropriate acclimatization and gentling period.

Rats injected with pyrogen-free saline and exposed for 90 minutes at an average power density of 5 mW/cm^2 did not display a significant increase in colonic temperature compared with saline-injected, sham-irradiated controls (Fig. 3). A slight but significant difference ($P < 0.05$, Student's t-test) was observed in the colonic temperature of saline-injected rats exposed to 10 mW/cm^2 compared with that of sham-irradiated rats. Therefore, increases in colonic temperature observable in the endotoxin-treated hypothermic rats cannot be measured in rats with an intact thermoregulatory system under low-intensity (≤ 5 mW/cm^2) microwave radiation.

The effect of ambient temperature on the magnitude of endotoxin-induced hypothermia is shown in Figure 4. An ambient temperature-dependent response of the colonic temperature is evident. The colonic temperature of saline-injected rats at an environmental temperature of 22 °C is close to that of endotoxin-injected rats exposed to microwaves at a power density of 10 mW/cm^2 and saline-injected, sham-irradiated rats (Fig. 3). At this power density, the colonic temperature of the endotoxin-treated rats fell between that of rats held at ambient temperatures of 26 °C and 30 °C, which delimit the thermoneutrality zone for the rat [Ederstrom, 1966]. At ambient temperatures above 30 °C, a febrile response to endotoxin by rats occurs [Szekely and Szelenyi, 1979]. Consequently, at power densities above 10 mW/cm^2 or at ambient temperatures above 30 °C, increases in core temperature would result from the animal's pyrogenic response to endotoxin as well as from the increased thermal load from microwaves or elevated ambient temperature.

DISCUSSION

The exposure of male albino rats injected with bacterial endotoxin to continuous-wave microwave radiation (2450 MHz) at an average power density of 1 mW/cm^2 was associated with a significant elevation of body temperature compared with that of endotoxin-injected, sham-irradiated rats. The elevation in body temperature was related to the power density (10 mW/cm^2 > 5 mW/cm^2 > 1 mW/cm^2). Saline-injected rats exposed

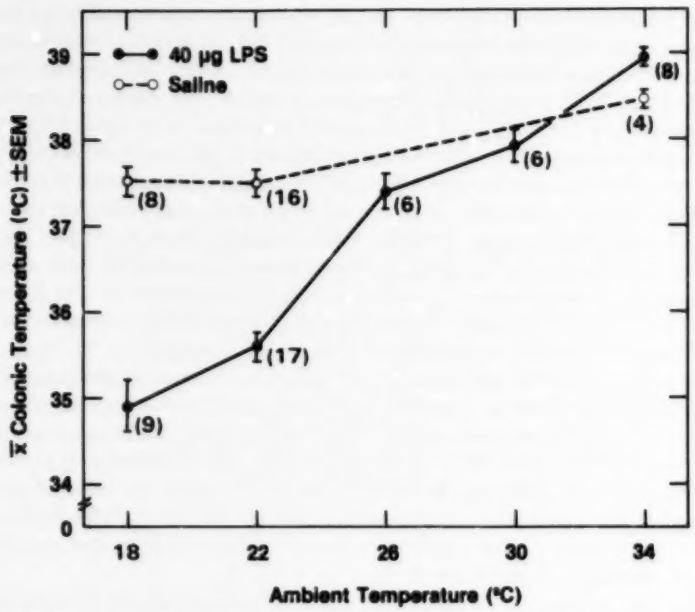

Fig. 4. The effect of ambient temperature on colonic temperature of rats 90 minutes after treatment with endotoxin. The number of animals per treatment is shown in parentheses.

TABLE 1. Temperature Response of Endotoxin-Treated Rats Exposed to Microwaves at 0.5 mW/cm² Following Two Weeks of Procedural Acclimatization

Treatment	Mean colonic temperature (°C) ± SEM		
	Initial	Final	ΔT
Sham-irradiated (N = 12)	37.6 ± 0.10	35.7 ± 0.20	−1.85 ± 0.16
Irradiated (N = 12)[a]	37.3 ± 0.10	36.3 ± 0.27	−0.99 ± 0.32[a]

[a] $P < 0.05$, Student's t-test.

at 0 or 5 mW/cm² did not show an increase in core temperature. In hypothermic rats the increase in the core temperature following microwave exposure agrees with findings of a study by Putthoff et al [1977] in which rats made hypothermic with cortisone displayed increased colonic temperatures following microwave irradiation. In both cases, thermoregulation was compromised by chemical means, and in both cases, microwaves caused an increase in colonic temperature.

For reasons not fully understood, the rat, unlike other species, exhibits a decreased body temperature following intravenous injection of endotoxin. It has been shown that the body temperature falls despite a concomitant rise in oxygen consumption at ambient temperatures of 20 °C or less [Kerpel-Fronius et al, 1966]. The absence of a rise in body temperature in rats following endotoxin treatment is believed to be due to a high rate of heat loss in these animals. This response may be related to the size of the rat, which has a large body surface area to mass ratio [Bligh, 1973]. Ambient temperature plays a critical role in this response to endotoxin; at temperatures above thermal neutrality, endotoxin-treated rats respond with a typical biphasic febrile response [Szekely and Szelenyi, 1979].

The microwave data (Fig. 3) do not show a linear relationship between power density and body temperature increase. At 1 mW/cm^2, a model comparable to a rat absorbing energy at a rate of 0.2 mW/g over a 90 minute period would display an increase in temperature of about 0.3 °C. Our results indicate a rise of approximately 0.8 °C. This excess is difficult to account for but may be due to the difference in pre-exposure temperatures between microwave- and sham-irradiated rats, the variability in the response of rats to endotoxin, the role that restraint-induced stress responses play in antagonizing the endotoxin response [Bligh, 1973; Berry and Smythe, 1964], the variability of heat resistance and thermoregulatory ability of rats [Wright et al, 1977], and/or the role that psychogenic stressors play in elevating body temperature [Justesen et al, 1974]. These factors may also account for the failure to observe a linear response in colonic temperature to increasing ambient temperature at and below 30 °C.

The administration of endotoxin to rats alters the thermoregulatory capacity of these animals and allows them to respond to changes in ambient temperature much like ectotherms. Under these conditions, small doses of thermalizing energy from low-intensity microwave irradiation were detectable as a significant increase of core temperature. In normal rats (saline-injected), the thermalization by microwaves at these low intensities must be compensated by an increase in the rate of heat loss. The similar effects on the body temperature of endotoxin-injected rats produced by microwave irradiation and by increases in ambient temperature indicate that generalized heating of the body was responsible rather than some direct interaction of microwaves with the nervous system. We have observed a similar response to low-intensity microwaves (2450 MHz) in mice made hypothermic with 5-hydroxytryptamine [Smialowicz et al, 1980]. Because of these findings, a more prudent assessment of the claims for "nonthermal" microwave-induced biological effects, that is effects observed in the absence of a measurable increase in temperature in animals with an intact thermoregulatory system, is warranted.

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DISCLAIMER

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